PULSATING SPLEEN IN MITRAL AND TRICUSPID DISEASES.[1]

By Morris Manges, M.D.,
PROFESSOR OF CLINICAL MEDICINE, UNIVERSITY AND BELLEVUE HOSPITAL MEDICAL COLLEGE; VISITING PHYSICIAN, MOUNT SINAI HOSPITAL, NEW YORK.

S. S., aged sixteen years, entered Mt. Sinai Hospital in November, 1910. She had been in the hospital one and a half years before on account of chronic endocarditis, and double mitral disease with auricular fibrillation, which was rheumatic in origin.

At that time it was noted that the liver was tender and pulsating, and that the spleen was also large and tender. She left the hospital much improved; but one month later the old symptoms came back, and since then there had been more or less loss of compensation, on account of which she reëntered the hospital.

The physical status was that of advanced double mitral lesions, with tricuspid regurgitation and a moderate dilatation of both ventricles. There was also a short localized diastolic murmur over the third space to the left of the sternum. An effusion in the right chest reached the angle of the scapula. The liver extended to the umbilicus; it was hard, nodular, tender, and pulsating. The spleen was also enlarged upward and downward, and almost extended to the umbilicus. It was hard and nodular; at the tip there was a large, firm nodule. The spleen reached almost as far as the liver, but it did not touch it, as a distinct space was felt between the two organs. No note was made of pulsation of the spleen at the time of admission.

The radial pulse was small, rapid, and irregular. The veins in the neck pulsated forcibly. The polygraph showed auricular fibrillation. Systolic blood-pressure was 110 to 115.

The blood count showed 6,700,000 to 8,000,000 red blood cells; 9000 to 13,000 white blood cells; 90 per cent. polynuclears; hemoglobin 84 per cent. The urine showed a heavy trace of albumin and many granular casts.

The peculiar feel of the liver and spleen led me to suspect syphilis, although no history of it could be obtained. The Wassermann reaction was strongly positive.

It was not until the patient had been in the hospital two weeks that the pulsation was observed in the spleen for the first time. It was probably present at the time of her admission, but it was not observed. It persisted throughout her stay in the hospital. It was a distinct expansible pulsation, which was best observed on bimanual palpation, its time usually preceding that of the liver, but sometimes it seemed to coincide with it.

The course of the disease was that of any ordinary advanced endocarditis, with an occasional infarct. She was in the hospital for a long time, and she left much improved, although when she left the fibrillation of the auricle and the pulsation of the liver and spleen were still present. Although efforts were made to trace her, she was never heard from again. This report has been delayed in the hope of observing the case again and also in the expectation of observing additional cases of pulsating spleen. However, in spite of systematic routine palpation of every enlarged spleen during the past six years I have been unable to find any additional cases.

---

[1] Read at the meeting of the American Climatological and Clinical Association, Washington, May 10, 1916.

It is to this feature of this case to which I would call attention, since this is a symptom of very rare occurrence. It was first described by Tulpius[2] in 1652. Gerhardt,[3] in 1882, reported a case in a man who had an old aortic regurgitation, and who also had malarial fever. Later on, Gerhardt[4] was also able to report two additional cases of aortic regurgitation, in both of which pericarditis with fever developed during their stay in the hospital. In both of these patients he discovered pulsation of the spleen during the febrile stage of the disease. He also noted that the increase in the size of the spleen was much greater than was usually observed under such conditions.

Prior[5] reported two cases. The first case was that of a well-compensated aortic regurgitation in which typhoid fever was contracted. The spleen only pulsated during the course of the fever. The second case was one of hypertrophy of the left ventricle, which was the result of overexertion in a workman, aged thirty-seven years. Pulsation of the spleen occurred during the course of an attack of croupous pneumonia.

Drasche[6] reported a case of pulsating splenic tumor in a female, aged seventeen years, with an advanced aortic regurgitation which had appeared during the febrile stage of an attack of croupous pneumonia. The enlarged pulsating spleen disappeared as soon as the fever was gone.

To these cases which have been collected by Litten[7] I have been able to add two additional cases, both observed by Gerhardt;[8] both of these men had aortic regurgitation and developed the pulsation of the spleen during attacks of lead colic; in both patients the pulsation disappeared as soon as the attack of lead colic was over. Fever was present in one patient and was absent in the other. In the former the liver also pulsated. No reference was made to the liver in the latter case.

[2] Quoted by Litten: Loc. cit. (7).

[3] Ztschr. f. klin. Med., 1882, Band iv, 449.

[4] Charité Annalen, 1887, Band xxii, 229.

[5] München. med. Wchnschr., 1887, Band xxxiv, 669.

[6] Wiener med. Blätter, 1888, Band xi, 1.

[7] Nothnagel's Spec. Pathol. u. Therap., 1898, Band viii, Theil 2, p. 75.

[8] Centralbl. f. klin. Med., 1888, Band ix, 1.

With the exception of the historic case of Tulpius, which he observed in 1652, all of these cases were observed within the period from 1882 to 1888. I have been unable to find any cases after this date; a striking commentary on how the careful clinical examinations have been lost since the laboratory has played such a preponderating role in the making of diagnoses.

In all of these cases the splenic pulsation was arterial and was synchronous with the cardiac systole and increased and diminished with the vigor of the cardiac action. Prior's 2 cases showed this very strikingly; in the pneumonia patient the pulsation seemed very much less during the period of cardiac weakness; and it also almost disappeared as soon as the spleen decreased in size when the fever was gone. In the 2 cases of lead colic it was only present during the period of very high blood-pressure associated with the colic.

According to Gerhardt and Litten three factors are needed: an enlarged spleen, chronic cardiac disease, and an acute febrile disease. The chronic cardiac disease always represented a marked hypertrophy of the left ventricle, usually with aortic regurgitation; only once was there idiopathic hypertrophy of the left ventricle. The fevers included pneumonia, malaria, and typhoid fever. Litten explains the phenomenon as being the result of the fever increasing the vascularity of the spleen and relaxing its tissue. As a result, the pulsation of the arteries is transmitted more readily and distinctly through the organ. This explanation does not hold in the 2 cases of lead colic. Here Gerhardt assumes that a high blood-pressure associated with the lead colic makes the pulsation in the splenic vessels more forcible.

The present case differs from those thus far reported. There was no evidence of aortic regurgitation, unless the short diastolic murmur in the third left space might be accepted as evidence of aortic regurgitation, of which, however, there was no other symptom, and there was no Corrigan pulse or double tone in the vessels; the tracings showed no sign of it; the hypertrophy of the left ventricle could easily be explained by the mitral lesion.

How can it be explained? Surely not according to Gerhardt and Litten, for most of the conditions postulated by them are absent; there was neither fever nor acute infectious disease, and there was no aortic regurgitation. There was an hypertrophy of the left ventricle and there was an enlarged spleen. The hypertrophy of the left ventricle was mitral in origin; the splenic enlargement was partly due to the cardiac disease and partly to the syphilis. There was no evidence of aneurysm or any pulsating vascular tumor.

The question at once arises whether the pulsation may not have been transmitted from the heart, liver, or aorta. This may be dismissed because the pulsation was distinctly expansile and independent of any of the other viscus. This could readily be demonstrated on bimanual examination. There were no pulsating tumors or cysts of the spleen, and the pulsation must have originated in the splenic vessels. This was well illustrated by a series of tracings which was made by Dr. Alfred E. Cohn. The tracings include the time relations of the spleen to the liver, cardiac apex, and radial and femoral arteries. These tracings were studied by Dr. Cohn and Dr. B. S. Oppenheimer. It is not an easy task to draw conclusions from them, as unfortunately no jugular or abdominal aorta tracings were made. Dr. Cohn was inclined to regard the splenic pulsations as arterial. The pulsations in the liver he considers to be of the ordinary ventricular type. Dr. Oppenheimer, after a very careful study of the tracings, is unable to reach any positive conclusions concerning the nature of the splenic tracings. He agrees

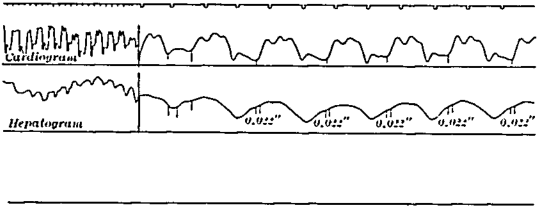

FIG. 1.—Tracings of heart and liver.

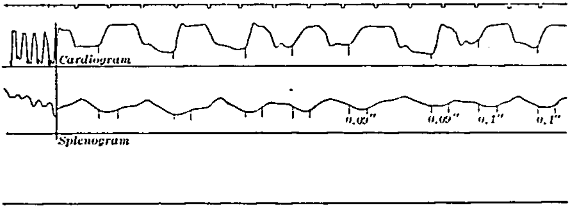

FIG. 2.—Tracings of heart and spleen.

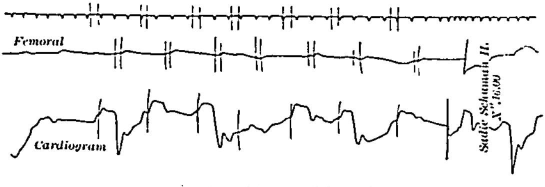

FIG. 3.—Tracings of heart and femoral artery.

with Dr. Cohn in considering the liver pulsations to be ventricular. When two observers who have had such a large experience in interpreting tracings are unable to agree, I believe it will be wisest to simply state the findings and to leave their exact determination to the future, when possibly more cases will have been observed. The following are the time relations:

1. The cardiohepatic time is 0.022 second (Fig. 1).
2. The cardiosplenic time is 0.09 or 0.1 second (Fig. 2).
3. The cardiofemoral time is 0.1 second (Fig. 3).

These figures would indicate that the splenic pulsations are arterial, as the time relation of the femoral artery agrees so closely with that of the spleen. The dynamic conditions which exist in this case, however, do not warrant this assumption, as this conclusion would predicate the existence of an aortic regurgitation, of which there are almost no evidences. The only possible warrant for it is the short diastolic murmur in the third left space. This is not enough for a diagnosis of aortic regurgitation, all the evidence of which was absolutely lacking, although it was carefully sought for during the long period she was under observation. But even if we did assume it to be present, all the other postulates of Gerhardt and Litten are absent. To assume that the pulsation in the spleen is arterial and that that in the liver is venous is an improbable assumption. The most plausible explanation is to attribute both pulsations in the liver and the spleen to the same source, *i. e.*, venous, for this is the type usually seen in tricuspid regurgitation. This, too, is improbable, for it is inconceivable that the propagation of the pulsations in the liver which is so closely related to the inferior vena cava should also be propagated into the splenic vein, which is a branch of the portal vein.

It is greatly to be regretted that I cannot offer any explanation of this unique case. Unfortunately, it is the only case of the kind which has ever been reported, and it was observed six years ago, when the new cardiographic methods were still in their infancy.

THE RATE OF EXCRETION OF THE THREE NITROGENOUS WASTE PRODUCTS, URIC ACID, UREA AND CREATININ IN NEPHRITIS, AS SHOWN BY COMPARATIVE STUDIES OF THE BLOOD AND URINE.

By C. K. Watanabe, M.D.,
NEW YORK.

(From the Laboratory of Pathological Chemistry, New York Post-Graduate Medical School and Hospital.)

The accumulation of the end products of protein metabolism in conditions of renal insufficiency is now a well-established fact.